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LETTER

Ambient Temperature Interferes to COVID-19 Ambient Temperature

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Angiotensin Converting Enzyme 2 (ACE2) is a blood pressure regulating enzyme that is attached to the outer surface in cells of the lungs, arteries, kidney, heart and intestines [1]. In addition to blood pressure regulating, ACE2 is involved in the pathophysiological processes by converting angiotensin isozymes during cell injury [2]. ACE2 converts Angiotensin II to Angiotensin-(1-7) [3]. ACE2 is also a receptor for some coronaviruses, including HCoV-NL63 (Human Coronavirus NL63), SARS-CoV (Severe Acute Respiratory Syndrome-associated Coronavirus) and SARS-CoV-2 (Severe Acute Respiratory Syndrome -associated Coronavirus-2), infectious agent of COVID-19 (Coronavirus Disease 2019) worldwide [4]. Many hypotheses have been made about COVID-19. Underlying diseases such as hypertension, diabetes and cancer, and environmental factors such as preventative methods and ambient temperature have been suggested to be involved in the outbreak of COVID-19. Because of the unclear treatment results and the panic following COVID-19, some news about prevention and treatment strategies have been quickly spread among people through virtual networks. One of these news is the effect of heat and ambient temperature on the contagion of SARS-CoV-2. This paper has surveyed the scientific possibility of intervention of ambient temperature on COVID-19.

Previous studies have shown that cells in the human body biosynthesize heat shock proteins when exposed to adverse environmental conditions such as low and high temperatures [5]. Heat shock protein contributes to the robustness and survival of cellular protein structures by its chaperone function [5]. Previous studies show that HSP72 (Heat Shock Protein 72) is expressed in human cells under changes of temperature out of physiologic ranges [5]. In regards to COVID-19, HSP72 increases the gene expression of ACE2 as SARS-CoV-2 virus receptor [4].

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On the other hand, ACE2 converts Angiotensin II to Angiotensin-(1-7) that affects phosphorylation and activation of AKT, AMPK and Sirt1 molecules through MAS membrane receptors [6]. Mentioned proteins activate the p-eNOS (Phospho-Endothelial Nitric Oxide Synthase) that increases intracellular NO (Nitric Oxide) [2, 3]. After binding the SARS-CoV-2 virus to ACE2, because of decreased ACE2 levels, Angiotensin II is not converted to Angiotensin-(1-7) (1 and 3). Follow of decreased Angiotensin-(1-7), nitrogen radical production and viral genome degradation will cease [7]. On the other hand, in ACE2 deficiency conditions, the cellular signaling between Angiotensin II and Angiotensin-(1-7) will be unbalanced toward Angiotensin II signaling [1]. Angiotensin II increases inflammation response by producing inflammatory cytokines and ROS (Reactive Oxygen Species), due to pneumonia and cell death [1].

In conclusion, staying in a hot environment, such as the dry and wet sauna or cold environment, like cold water pool for at least 15 minutes, can produce stress shock in the cells and gene expression of HSP72 (3 and 6). HSP72 increases the ACE2 gene expression being the receptor of SARS-COV-2 virus, inflammation, cell death and finally pneumonia [6]. Therefore, avoiding these heat and cold stress shocks is recommended to reduce the risk of SARS-COV-2 virus infection.

CONFLICT OF INTEREST

The author declares no conflict of interest, financial or otherwise.

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